the removal of the stones squeezed up out of the ureter and partly for the exploration of the ureter and calices.

Exploration of the ureter with the sound led to the detection of the stricture, a valve, which was excised through a longitudinal opening in the ureter at its seat. I consider this operation preferable to excision of the entire wall of the ureter and invagination after Van Hook's method, as it is followed by the least possible shortening of the canal. This point may be of importance if more than valvular stricture should have to be operated upon.

Exploration of the calices can be made just as well through an incision in a somewhat dilated pelvis when the latter is wide enough to permit exploration with the finger and sound. For additional security against leaving stones in hidden calices with narrow entrances it would be well to explore through the convex surface of the kidney with steel needles. When this is done in addition to digital exploration through the pelviotomy-wound I think there is as little risk of overlooking stones as exists when the convex surface of the kidney is divided. To avoid dividing the posterior surface of the kidney avoids hemorrhage, the operation on the pelvis of the kidney being entirely bloodless.

For the local treatment of pyelitis—that is, inflammation of the pelvis and calices—drainage through the wound in the pelvis will be sufficient, at least in ordinary cases of stones and valves in which, by removal of the cause, we may reasonably expect the pyelitis to come to an end.

269 LA SALLE AVENUE.

A CASE OF SYRINGOMYELIA LIMITED TO ONE POSTERIOR HORN IN THE CERVICAL REGION, WITH ARTHROPATHY OF THE SHOULDER-JOINT AND ASCENDING DEGENERATION IN THE PYRAMIDAL TRACTS.[1]

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IF one glances at the extensive literature on syringomyelia, as given in Schlesinger's recent monograph, he feels as though he must offer an apology for adding another case. Syringomyelia with arthropathy followed by autopsy and an examination of the spinal ganglia; ascending degeneration in the direct and crossed pyramidal tracts above the focus

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[1] Read before the American Neurological Association, June 4, 1896.

of destruction in the cord; gliosis extending from the second cervical segment to the extreme end of the conus terminalis; destruction of one posterior horn alone, from the second cervical to the first dorsal segment, with diminution of the lateral column on the same side; pachymeningitis involving a large portion of the dorsal region—these are conditions which seem to justify us in presenting the following case for your consideration:[1]

C. H., male, aged forty-five years, a native of Sweden, unmarried, and a laborer by occupation, was admitted to the nervous wards of the Philadelphia Hospital January 9, 1883. His family history did not appear to be especially significant. His father had died of phthisis, his mother of old age. Two brothers were living and in good health. His personal history previous to the present affection also failed to present factors of importance. He had had typhoid fever when twenty years of age, but no other illness of moment. Venereal history was denied, and he had always been temperate in his habits.

About three years before admission, while working in a sawmill, he did some heavy lifting and strained his back. He was obliged to go to bed for a day, but afterward felt no evil effects. About three years later he began to suffer from pains in the legs and a band-like pain about the lower part of the chest. These pains gradually grew worse, and soon his walking was interfered with. He noticed that his legs were getting weak, and that he could not walk as well as formerly. His feet would drag slightly and the toes would occasionally catch upon the ground and cause him to fall. He noticed also occasional cramp-like pains in the calves of his legs. Gradually loss of power became more and more marked, and when admitted to the hospital he was unable to walk, but could sit up in a chair.

The legs presented the symptoms of spastic paraplegia, and later on marked contractures made their appearance. Gradually loss of power became complete, and he lay in bed for the last four or five years with both legs drawn upward toward the trunk, especially the right, the thighs being flexed more or less upon the abdomen and the legs flexed upon the thighs. He could not move the legs in the least, and rigidity was so great that it was impossible to extend them even passively. The knee-jerks were both markedly exaggerated. On the right side, however, the reaction gradually disappeared, owing to the fixed position of the limb. Ankle-clonus was also present, but was finally lost, likewise due to the fixation produced by the contracture. General muscular wasting also took place in both thighs and legs.

Early in the case the bowels were constipated, and he had also difficulty in voiding urine. Little by little paralysis of retention set in, and finally both bowels and bladder were emptied without the patient's knowledge.

Cutaneous sensibility was early lost in the legs; but the nature of the affection was not suspected until 1890, when a detailed examination revealed not only that the cutaneous sensibility was absolutely lost in both legs, but that this loss extended also upward upon the trunk anteriorly and posteriorly; upon the right side as far as the level of the nipple, and upon the left a little above the level of the umbilicus. The areas in which cutaneous sensibility was lost and those in which it was preserved were not sharply demarcated. In examining him with the æsthesiometer from below upward his answers were at first uncertain as the levels mentioned were approached, and only when these levels were fairly past did his answers become positive. In all portions of the body, save the legs and the portions of the trunk already mentioned, tactile sensibility was well preserved. It was found, however, that the sense of temperature was absolutely lost over the right arm, the right shoulder and the right side of the neck, and also upon the adjacent portions of the trunk above the nipple-line. There was also some analgesia of the right arm, though this was not absolute (see Figs. 1 and 2). The band-

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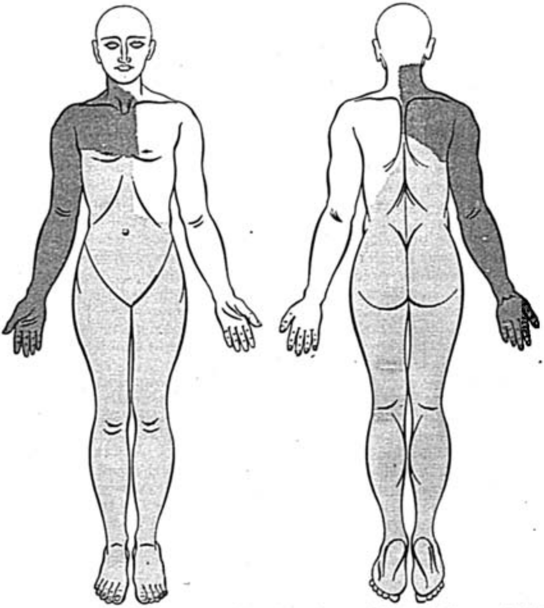
[1] See also clinical lecture delivered at the Philadelphia Hospital, 1890, by one of the writers. International Clinics, second series, vol. ii. p. 108.

FIG. 1. FIG. 2.

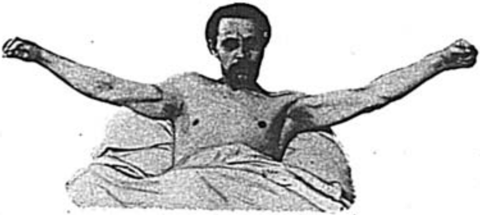


In the areas shaded lightly there was loss of sensation to all forms. In the areas shaded heavily there was absolute loss of the temperature-sense and a decided impairment of the pain-sense. In the unshaded areas sensation was normal.

like pains upon the chest noted early in his case steadily persisted, and were referred more especially to the fourth, fifth, and sixth intercostal nerves upon the left side.

In 1888 it was noticed that the right shoulder had become swollen. Examination of the joint revealed that the joint-surfaces were much roughened, and also that some effusion had taken place into its cavity. No pain was present at this time. For a time the effusion steadily increased, and finally rupture of the capsular ligament with infiltration of the subcutaneous and intermuscular connective tissue took place. A cellulitis resulted, which presented the ordinary symptoms of swelling, redness, and local heat, but with a very slight degree of pain. After a number of weeks the effusion was again absorbed, and it was then noticed that when the patient attempted to extend the right arm that the humerus assumed the position of a subglenoid luxation. (See Fig. 3.)

FIG. 3.



From a photograph taken in January, 1890, showing the arthropathy of the right shoulder.

At various times the effusion into the joint recurred, and at such times a pouch-like dilatation of the capsule would distend the axilla. Little by little the humerus shortened, considerable destruction of bone taking place.

In addition to the above facts the patient presented nothing of importance, save that on several occasions he developed small abscesses —one, for instance, over the left ilium and over the left gluteal region. Bedsores also made their appearance at various times and persisted until the end. Mentally he was extremely irritable, ascribing his affection to an overdose of arsenic, which he claimed had been administered to him by a resident physician in mistake.

The examination of the blood revealed nothing of importance. An examination of the eyes by Dr. de Schweinitz was negative.

Up to about two years ago the patient's general condition remained fairly good; but little by little emaciation set in, and he finally died with symptoms of exhaustion on the 14th of December, 1895.

Autopsy, December 14, 1895. Body much emaciated. Emaciation general; no local atrophies. Both legs contracted, the right leg abducted and semiflexed, the left leg adducted and partly flexed. A large, gangrenous bedsore covered the lower two-thirds of the sacrum. A similar bedsore was also noted over the right tuberosity of the ischium and one over the right trochanter.

Brain. Scalp unusually thin. Calvarium normal, save perhaps that the diploic structure was less pronounced than usual. Pacchionian bodies small in number. External layer of the dura normal; its internal layer smooth and whitish-gray in color. Veins of the pia not distended. The cerebro-spinal fluid about normal in amount. The brain as a whole was softer than normal. There was, however, no local softening, the latter being everywhere diffuse. The cerebellum seemed slightly firmer than the cerebrum. The meshes of the pia and arachnoid were somewhat œdematous. The pia was readily detached from the cortex. Vessels of base rather small, otherwise normal, except here and there an atheromatous patch. Ventricles not dilated; their walls rather pale. Velum interpositum normal, its veins not distended. Choroid plexuses normal and but slightly cystic. Foramen of Monro normal, not dilated. Walls of the third ventricle slightly pale. On section the brain seemed moist, but revealed very few puncta vasculosa. There were no lesions of the basal ganglia or capsules. The crura were normal on section. Pons and medulla likewise normal. No dilatation of aqueduct of Sylvius nor of central cavity in lower portion of medulla.

Spinal Cord. The dura of the cord was adherent almost in its entire thoracic portion. The substance of the cord was very soft, especially in the dorsal region. In this region also the dura mater was much thickened.

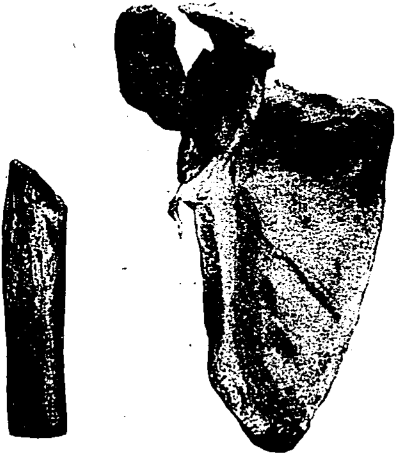
Thorax. Left pleura free from adhesions. About two ounces of fluid in pleural cavity. Right pleura extensively adherent; adhesions firm and difficult to break. Lungs crepitant. Right lung somewhat œdematous in lower lobe, but pieces of the latter floated readily. Some frothy mucus in bronchi. No tubercular deposit. Bronchial lymphatic glands small and pigmented. The pericardium was smooth and contained a normal amount of fluid. Heart-muscle pale and flabby. Both ventricles distended with dark fluid blood. No lesions of heart-valves; no atheroma of aorta.

Abdomen. On opening the abdomen a large amount of sero-purulent fluid, estimated at about thirty ounces, escaped. The omentum was very thin, containing little or no fat. It was adherent to the small intestine. The small intestine and the ascending, descending, and transverse colon were matted together by extensive old peritoneal adhesions. The viscera were everywhere covered by extensive exudate, apparently old. The stomach was distended, a large amount of yellow chyme escaping from the œsophageal opening. The mucous membrane was exceedingly pale, presenting here and there minute patches of erosion. These erosions were present both on the anterior and posterior walls.

The spleen was normal in color and consistence, but small. The liver was evidently diminished in size; capsule not adherent; cut surface distinctly paler than normal. The kidneys presented adherent capsules and fatty changes, especially in the cortex, which was also here and there a little thinner than normal.

On examining the right shoulder-joint it was found that the proximal end of the humerus was in the position of subglenoid luxation, and that it was freely movable and could be readily reduced. On opening the joint it was found that its capsule was much thickened and the inner surface of the latter much roughened. The head of the humerus had disappeared, the bone having been eroded to some little distance below the surgical neck. The surface of the glenoid cavity was relatively smooth, but the adjacent portions of the capsular ligament were festooned with vegetations. Here and there it was villous. A cystic tumor was also found in the axilla containing a friable fatty material. The cavity of this tumor communicated with the cavity of the joint by a small sinus. The scapula, a portion of the humerus, and portions of the capsular ligament were removed. Subsequently they were prepared, and are herewith presented. (See Figs. 4 and 5.) The humerus presents complete loss of the head and surgical neck, the proximal end being represented by a porous surface extending obliquely upward and outward. Adjacent portions of the shaft are also much roughened and porous. The scapula likewise presents extensive changes. The surface of the glenoid cavity has been much eroded, and has become roughened and porous; but this is neither the most interesting nor the most striking feature. The glenoid surface seems to be abnormally large, and upon examination it is seen that extensive bony deposit has taken place along its edges. This deposit is especially marked upon its posterior and inferior edges. The latter is curiously festooned. Immediately back of the posterior portion of the glenoid cavity other extensive deposits are noted. The coracoid process also exhibits marked changes. Not only is it thickened, roughened, and porous, but it exhibits around its entire edge a thick and firm accretion, averaging at least a half-inch in width. The latter is separated from the coracoid itself by a well-marked line of demarcation.

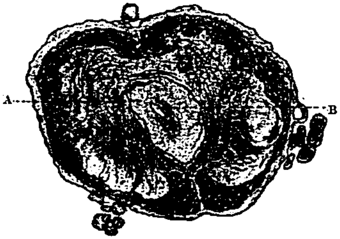
FIG. 4. FIG. 5.



Humerus and scapula of the right side. The head of the humerus has been destroyed. The glenoid surface of the scapula is enlarged, and its edges are thickened by bony deposit. The coracoid process presents peripheral accretions.

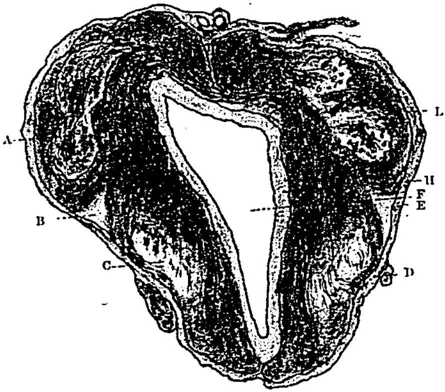
The central nervous system, with portions from several nerves, was placed in Müller's fluid, and sections were made from levels corresponding to nearly every spinal root, and stained with carmine, Delafield's hæmatoxylin, and by the methods of Weigert, Pal, and Marchi. Numerous sections were also made from different spinal ganglia. The microscopic work was done in the Pepper Laboratory.

FIG. 6.



Section from extreme end of conus. A, tumor-like mass broken down in the centre at B.

FIG. 7.



Section from the level of the fifth lumbar roots. A and L, anterior horns; B and E, degenerated crossed pyramidal tracts; C and D, posterior horns; F, cavity occupying the area of the posterior septum; H, gliomatous tissue.

Conus Terminalis. (See Fig. 6.) Sections from the extreme end of the conus show a tumor-like mass occupying the centre of the cord and containing a small central cavity. The borders of the gliosis are well defined by fibres, colored black by the method of Weigert. The tissue bordering the cavity appears looser in structure than that more peripheral, and shows a tendency to a radiated structure. The nuclei of the neuroglia-cells are more abundant toward the periphery of the mass. The posterior columns are very slightly involved and only at their anterior portion.

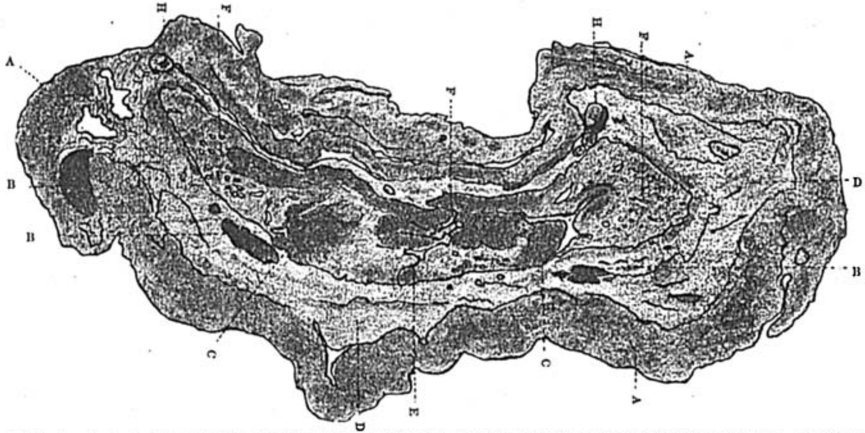
Upper Part of Sacral Cord. The cavity occupies a greater portion of the tumor, which involves the anterior part of the posterior septum and both anterior horns, especially the left. The vessels at the border of the gliosis are somewhat sclerosed. The cavity appears almost as if formed by the removal of a portion of the tissue by the knife in cutting the sections, owing to the absence of any thickening in the growth at this part.

The central canal is not involved in the tumor, but a mass of proliferated cells is found in the area usually occupied by it. This condition is well known in normal cords. In some sections at this level the canal is found patulous and lined by an unbroken layer of cylindrical cells. The anterior and posterior roots show no change; indeed, the posterior are normal throughout the cord in their extramedullary portion, whereas in a part of the thoracic cord the anterior are degenerated.

Fifth to Second Lumbar Segments. (See Fig. 7.) The cavity now extends along the posterior septum almost to the pia mater. The cells of the anterior horns are numerous. The crossed pyramidal tracts are degenerated. The central canal is patulous at the level of the fourth and third lumbar segments and lined with ependymal cells, and is anterior to the tumor. The gliosis does not alter its shape essentially until at the level of the second lumbar segment, in which region it is no longer triangular with its apex at the junction of the periphery with the posterior septum, but is irregular in shape and occupies a large part of the cord, which contains little normal tissue. The pachymeningitis makes its appearance at the second lumbar segment and extends to the exit of the third dorsal roots.

Exit of Eleventh to Exit of Fourth Thoracic Roots. (See Fig. 8.) It was impossible to count the roots at their junction with the cord except by tearing away the thickened membranes; we have, therefore, been obliged to count the segments according to the points of exit of the roots from the dura. There are round-cell infiltration and proliferation in the greatly thickened membranes, even the spinal roots are involved, but within the thickened meninges the fibres of the posterior roots are found apparently normal; the anterior are much degenerated at the level of the exit of the fourth thoracic roots. The posterior horns are especially involved in the destruction of tissue. Very few bands of normal nerve-fibres are found within the cord, which is almost entirely replaced by the gliosis and cavity. The cord is closely united with its membranes. The vessels of the meninges and cord are thickened and show hyaline degeneration; many are surrounded by a clear zone faintly colored by the carmine. The pachymeningitis at the exit of the fourth thoracic nerve is more confined to the posterior part of the cord. The cavity extends to the pia through the posterior horns in sections where the pachymeningitis is most marked. The endothelial layer between the dura and the arachnoid shows the proliferation described by Köppen. The only hemorrhages which can be noticed—small and few in number—are in the loose, fatty connective tissue external to the dura. In some sections the vasa vasorum are enlarged about a small artery in the cord. Abundance of amyloid bodies may be seen. The pia is thickened both above and below the parts where the dura has again become normal.

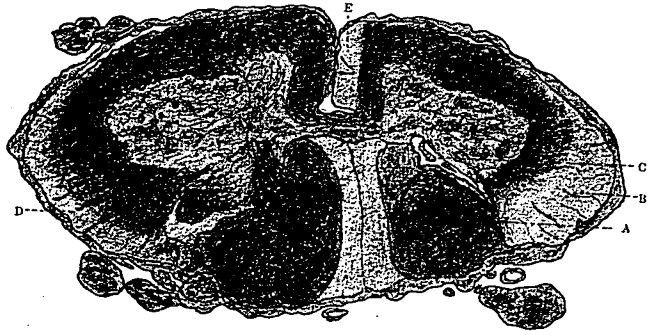
FIG. 8.



Section from the level of the exit of the eighth thoracic roots. The dura, pia, and arachnoid are greatly thickened and united. The cord is flattened and entirely degenerated except at C C, where a few medullated fibres are still to be seen. Scattered medullated fibres may also be found in a few other regions. Gliomatous tissue occupies a large portion of the cord, and in certain places, as at E, is broken down and causes cavity-formation. A. Thickened dura. B. Posterior roots not degenerated, a few fibres may possibly be found not perfectly normal, but these are rare. C, C. Masses of medullated fibres in cord. D. Thickened arachnoid and pia. F. Posterior septum containing a cavity. F, F. Thickened bloodvessels in the lateral columns. H. Greatly degenerated anterior roots. I. Anterior fissure.

Level of First and Second Thoracic Segments. The cavity shows a tendency to become limited to the right posterior horn, noticeable in the second thoracic segment and especially in sections from the first thoracic; in the cervical region it does not extend to any other part of the cord. The walls of the cavity are formed by a slight thickening of the neurogliar tissue, but the dense structure seen in sections from lower segments no longer exists. The cavity appears more as a tear in the normal tissue, with a slight band of thickened neuroglia containing numerous deeply stained nuclei along a portion of its walls. Considerable degeneration is noticed in both lateral columns, especially in that of the right side, and also in the right anterior column along the anterior fissure. The left anterior column is apparently normal. This degeneration is due to destruction of tissue lower down. The pia is very much thickened. The peripheral zone of neuroglia (peridym of v. Lenhossék) shows the thickening described by v. Lenhossék in all chronic spinal processes. The sclerosis involves the direct cerebellar tract and Gowers's column on each side of the cord. The right column of Goll and the anterior part of the left are destroyed as high as the first thoracic segment. Above this region the right lateral column is not occupied by the cavity or gliosis except in the form of secondary degeneration. We employ the term gliosis as distinctive from gliomatosis in the sense given by Hoffmann.

FIG. 9.



Section from the level of the eighth cervical roots. The entire right side of the cord is smaller than the left. A. Contracted posterior horn. B. Crossed pyramidal direct cerebellar and Gower's tract degenerated upward. C. Cavity limited to the posterior horn. E. Ascending degeneration in the area of the right pyramidal tract. D. Degeneration in the direct cerebellar and Gower's tract of left side.

Cervical Cord (See Fig. 9) *and Medulla.* The columns of Goll are entirely degenerated, and extend laterally at the eighth segment along the posterior commissure in a flask-shaped form; this appearance is not long preserved, but the degeneration in these columns does not withdraw from the posterior commissure. The degeneration in the left lateral column except along the periphery is not very marked, and cannot be traced very high; in the right lateral column it is more noticeable and may be traced into the substantia reticularis as high as the second cervical segment, decreasing gradually in intensity. This degeneration is not closely confined to the area usually considered as occupied by the fibres of the crossed pyramidal tract, although more intense in this portion. The degeneration in the right anterior column is very noticeable at the lower portion of the cervical cord, and may be traced in gradually decreasing intensity into the motor decussation, at which level it has almost ceased. It is chiefly located along the right border of the anterior fissure.

In the right lateral column in the eighth segment a few swollen axis-cylinders are noticed, and by the method of Weigert most of the fibres which are preserved are seen to be of small size. Such fibres are not observed in the degeneration of the columns of Goll, which contain only a very few medullated fibres. There is a semi-circular band of degenerated tissue in the right column of Burdach, seen only in the eighth segment, which passes from the neck of the posterior horn with the concavity outward and joins the degenerated columns of Goll about halfway toward the periphery. In this band these small nerve-fibres may also be found. It has not exactly the location of Schultze's comma-zone. The right posterior horn is very small, and is replaced antero-posteriorly by the cavity, which extends as high as the second cervical segment. There is in all cervical sections a zone of sound tissue between the median end of the cavity and the central canal. The degeneration along the periphery of each lateral column in the area occupied by the direct cerebellar tract and the column of Gowers, a little more intense on the right side, extends beyond a line drawn through the anterior part of each anterior horn, but decreases in intensity in ascending and withdraws toward the posterior horn. The right posterior roots appear normal in their extramedullary portion, but only very few fibres are found, and these merely in certain sections, entering the right posterior horn. They can be traced only a very short distance. In the lower cervical region the whole right side of the cord is smaller than the left, especially noticeable in the right lateral column, and the right anterior horn contains fewer fibres, probably the result of loss of fibres from the right posterior roots. The ganglion-cells appear normal. The lateral limiting layer (Grenzschicht of Flechsig) is not degenerated in any part of the cervical cord, probably because this zone contains short associative fibres. In the second cervical segment a narrow band of neurogliar tissue is found in the right posterior horn, corresponding to the location of the cavity below. This band is rich in cells with deeply stained nuclei, a condition which agrees with the statements of Hoffmann[1] that the gliosis is especially rich in cells where it is progressive. The right posterior horn is very narrow, and does not become broad at any part before its final disappearance. The sclerosis corresponding to the columns of Gowers cannot be traced apart from that of the direct cerebellar tracts. It becomes less in degree and finally disappears in the restiform bodies. At the second cervical segment there is a partial separation of the two tracts on the right side, but this is not apparent in sections from a higher level. This decrease in intensity would seem to indicate that

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[1] Hoffmann: Deutsche Zeitsch. f. Nervenheilk., 1892, vol. iii. p. 85.

the direct cerebellar tracts receive fibres in the cervical region. The degeneration in the columns of Goll does not go beyond the nuclei of these columns. The columns of Burdach are not degenerated in the cervical cord; there is great destruction in the right column of Burdach at the first dorsal, and yet from the seventh cervical segment no degeneration can be seen in these columns, confirming the statements that in the cervical region they are chiefly formed by fibres from the upper extremities.

There is no degeneration in the anterior pyramids and none in the fillet. Had the cavity in the right posterior horn extended a little higher there would have been bulbar symptoms added without paralysis in the upper extremities.

The Ascending Degeneration. Ascending degeneration in the lateral columns has been known for a long time, but forgotten until within the last few years. Klippel and Durante[1] give the limited literature on this subject up to 1895. Probably the first clear statement of this condition is to be found in the thesis of Michaud,[2] published in 1871, in which the following words may be found, which, when translated, read as follows: "It is not uncommon to find an ascending sclerosis in the lateral columns above the lesion; but, as a rule, it soon becomes indistinct and does not extend to a great height."

In May, 1893, Williamson[3] described degeneration of the direct pyramidal tracts in the upper thoracic and lower cervical regions which extended about four inches above a focus of transverse myelitis to the junction of the upper with the middle third of the cervical cord. The crossed pyramidal tracts were not involved above the lesion. He considered it probable that a localized spot of myelitis had at one time involved the direct pyramidal tracts, and that the degeneration was to be explained by the well-known law of Waller. Sottas[4] (November 25, 1893) reported four cases of spinal syphilis in which the crossed pyramidal tracts showed an ascending degeneration beginning in the thoracic region and extending in gradually diminishing intensity into the cervical. He gave this the name of "retrograde degeneration." He traced the sclerosis to its termination in the cervical cord, stated that it could not be due to a lesion higher up, emphasized the fact that the intensity of the process diminished in proportion to the distance of the section from the focus, and compared the condition to the degeneration of the central end of cut peripheral nerves.

In 1894 Raymond[5] reported ascending sclerosis of the crossed pyramidal tracts, and Gombault and Philippe[6] four cases, in one of which the direct pyramidal tracts were also affected. In two of their cases this was due to syringomyelia. In 1896 Dejerine and Sottas[1] have added one more case of ascending degeneration in the direct and crossed pyramidal tracts. According to Dejerine and Sottas, ascending degeneration has also been reported by Werding, Schmauss, and Daxemberger.

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[1] Revue de Médecine, 1895, p. 598.

[2] Méningite et myélite dans le mal de Pott. Thèse de Paris, 1871.

[3] British Medical Journal, 1893, vol. i. p. 946.

[4] Comptes Rendus des séances de la Société de Biologie, November 25, 1893.

[5] Arch. de Neurologie, February, 1894, Case III.

[6] Arch. de Médecine Expérimentale, 1894.

We can find no case in which this degeneration was unilateral in the spinal cord, as in the case we now report. The direct pyramidal tract was involved in the cases of Williamson, Gombault and Philippe, Dejerine and Sottas, and in ours.

James Hendrie Lloyd[2] (March, 1893), in his description of a case of syringomyelia, mentioned particularly that he had observed degeneration of one pyramid in a section taken above the termination of the cavity, and gave a picture of the section.

Hatschek[3] describes degeneration of both pyramids in a case of syringomyelia, which he does not believe to be of the ordinary descending type.

Schlesinger,[4] who has studied the literature of syringomyelia so carefully, states that Schultze, Rossolimo, Hoffmann, and Miura have mentioned a similar condition. In Schlesinger's cases, from lesion of the oblongata, the degeneration was more intense in the cerebral portions of the pyramids than in the spinal, owing to the presence of fibres in the former, which do not descend into the cord. He considers this degeneration of the pyramids as retrograde.

In our case there can hardly be any doubt of the existence of this ascending (cellulipetal, retrograde) degeneration in the right lateral column, involving the area of the crossed pyramidal tract especially, and extending from the upper thoracic region to the substantia reticularis of the second cervical segment, and in the right anterior column from the upper thoracic region to the motor decussation. The degeneration in the left lateral column does not extend very high. We base our conclusions on the following facts:

1. Absence of any macroscopic lesion above the medulla.
2. Degeneration of the crossed and direct pyramidal tracts on the *same* side of the cervical cord, intense in the lower cervical region near the focal lesion and diminishing gradually in intensity in the cervical segments, and finally becoming very indistinct in the upper cervical region.
3. Absence of all degeneration in the anterior pyramids.
4. Long duration of a chronic disease.

Owing to the kindness of Drs. Dejerine and Sottas, we have examined the preparations of the case published recently by them, and must consider that the degeneration we describe is not unlike theirs, although almost entirely unilateral. It is Dr. Sottas who has made this retrograde degeneration prominent. In our case the crossed pyramidal tract contains many fine, possibly atrophied, fibres and some few swollen axis-cylinders, and the sclerosis is by no means so great as in the cellulifugal degeneration of the columns of Goll. This cellulipetal degeneration in motor nerves and motor roots has been known for some time (see *Traité de Médecine*, tome vi. p. 662, and case reported by Dejerine and Spiller[1]).

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[1] Archiv de Physiologie, January, 1896.

[2] University Med. Mag., March, 1893.

[3] Wiener med. Wochenschrift, 1895, p. 1154.

[4] Arbeiten aus dem Institut. für Anatomie u. Physiologie, Obersteiner, Heft iv. 1896.

In our own cases certain fibres of association may be considered degenerated, but attention must be called to the fact that it is the area occupied by the direct and crossed pyramidal tracts which is chiefly affected, whereas associative fibres are found in the entire antero-lateral column. The sclerosis is probably due in greater part to atrophy of motor fibres deprived of their function, as it has been noted in cases only of long duration. The less involvement of the left side of the cord in this retrograde degeneration may be explained by the supposition that the right side of the thoracic portion of the cord was affected before the left, and that the process is therefore older on this side. The limitation of the gliosis to the right posterior horn in the cervical cord and the predominance of the clinical phenomena in the right lower limb some years before death render this view probable.

Columns of Gowers and Direct Cerebellar Tracts. The sclerosis in these tracts, more especially on the right side, is intense, and yet it is impossible to separate one of these from the other in the medulla oblongata and to trace the degeneration of Gowers's tract into the pons, as has been done by v. Monokow, Flechsig, Loewenthal, Auerbach, Mott, Tooth, Patrick. By some of these the tract was followed much further. Gombault and Philippe[2] were equally unable to separate the degeneration of these two tracts. In our case the sclerotic area gradually diminishes in intensity and withdraws toward the posterior horn, and finally disappears in the inferior cerebellar peduncle. Gombault and Philippe have recorded a similar diminution of the sclerosis in the direct cerebellar tracts, which they consider an anomaly. We are rather disposed to believe that the direct cerebellar tract receives fibres within the cervical cord as well as within the dorsal. Scattered cells corresponding to the location of the columns of Clarke have been noted in the cervical region. The sclerosis in our case may be from direct lesion of the fibres of these tracts or of the cells of the gray matter supplying the tract of Gowers and of those in the columns of Clarke. It cannot be a mere thickening of the peripheral neurogliar tissue described by v. Lenhossék[3] as common in all chronic spinal processes.

Pachymeningitis. This case shows intense pachymeningitis from the second lumbar to the exit of the third thoracic roots. This explains the pain felt in the fourth, fifth, and sixth intercostal nerves of one side. In most cases of syringomyelia the pia and dura are normal, but Hoffmann has found more than a dozen cases with thickening of the spinal membranes, usually in the cervical region. Joffroy[1] claims that in pachymeningitis cervicalis hypertrophica the inflammation penetrates into the cord from the membranes. Rosenblath,[2] Ziegler,[3] and others entertain a similar opinion. Hoffmann considers the pachymeningitis secondary to the syringomyelia. Wieting[4] and Köppen[5] object to the title of pachymeningitis for the form described by Charcot, and claim that the cord and membranes are involved simultaneously.

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[1] Comptes Rendus des séances de la Société de Biologie, 1895.

[2] Loc. cit., p. 568.

[3] Der feinere Bau des Nervensystems, p. 247.

In our case the pia is thickened where the dura is normal, which points perhaps toward an origin in the cord, and the intensity of the fibrous thickening and of the vascular changes in the membranes corresponds to the location of the greatest alteration in the cord and its vessels. At this part also the posterior horns are much affected. According to Hoffmann, the posterior horn is the part by which the cavity usually reaches the pia. The pachymeningitis is only in the thoracic region, and while the limitation of this process to the cervical cord has been shown by Adamkiewicz,[6] Wieting, Bouchut,[7] Köppen, etc., to be incorrect for some cases, still the normal condition of the cervical membranes in our case, where the cavity is very small and the intense involvement of the thoracic portion where the syringomyelia is most prominent, must be regarded as somewhat atypical for primary pachymeningitis, and as indicating a simultaneous affection or one secondary to the cavity-formation in the cord. Other cases are reported in which the pachymeningitis was situated where the cavity-formation was most intense, as in Rosenblath's case, No. IV.

We are inclined to believe that in our case the two processes were simultaneous, although there are certain conditions which seem to indicate possibly a primary location in the cord.

The vertebral canal is considerably narrower in the thoracic region than in the cervical and lumbar; the cavity at this level occupied more of the cord than at any other part, and during life contained fluid which probably distended the cord considerably. Pressure must have been especially great here, and perhaps to this cause is due the pachymeningitis in the thoracic region.

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[1] De la pachyméningite cervicale hypertrophique. Paris, 1873.

[2] Deutsche Archiv f. klin. Med., 1893, No. 51, p. 210.

[3] Lehrbuch der speciellen path. Anatomie, vol. ii. p. 813.

[4] Ueber Meningomyelitis mit besonderer Berücksichtigung der Meningomyelitis cervicalis chronica. Inaug. Dissert., Marburg, 1893.

[5] Archiv f. Psychiatrie, 1895, xxvii.

[6] Pachymeningitis hypertrophica u. der chronische Infarct des Rückenmarks. Wien, 1890.

[7] Gazette de Hôpitaux, 1877 to 1879.

Arthropathy of Right Shoulder. We had hoped to be able to contribute something to the knowledge of these arthropathies, and with this end in view many of the spinal ganglia have been examined, but in the fifth cervical ganglion of the right side no very decided changes were found. It is true that the vessels were much distended, more so in the fifth and seventh ganglia of the right side than in any others. The sixth cervical ganglion of this side was not obtained. There were many cells with deeply stained nuclei (possibly a round-celled infiltration); some of the ganglion-cells had shrunken from their capsule, but the nerve-fibres appeared normal; fatty degeneration, intense pigmentation, marked cloudiness, deeply colored protoplasm, irregular nuclei, absence of nuclei, destruction of the entire cell, proliferation of the cells of the capsule and the occupation by these of the intracapsular space, decribed by Wollenberg[1] and Stroebe[2] in tabes, are absent. The spinal ganglia have not often been examined in syringomyelia. Holeschnikoff found no decided changes in them, and in Hoffmann's case, in which trophic changes in the fingers and arthropathy at the wrist-joint had been present, the spinal ganglia corresponding to these parts were found unaltered.

In the cord the cavity at the fourth, fifth, and sixth segments is located as in lower levels; we cannot, therefore, attribute this arthropathy of the shoulder to any different conditions existing at these parts. Darkschewitsch,[3] in discussing the synovitis of joints which occurs in the paralyzed limbs after hemiplegia, considers this as due to altered vascular circulation. According to his view, if one would explain this by hypothetical trophic nerves, he must assume that the same nerve-elements serve as trophic centres for the most different tissues which form the components of a joint. He will not decide which of the vasomotor fibres are affected. The vascular system is much involved in paralyzed limbs; we have only to refer to the œdema seen in such cases. Charcot, basing his opinion on four autopsies, believed that the arthropathies in hemiplegia are due to synovitis (quoted by Darkschewitsch). In the cases described by Darkschewitsch the enlarged, painless joint seen in tabes and syringomyelia does not seem to have been present. Fraenkel,[4] in a recent paper, has shown the frequency of vascular along with nerve-alterations in the trophic changes of tabes, neuritis, syringomyelia,ulcers, as presented by arthropathies, etc., and in experimentation in cutting certain peripheral nerves. He believes that the so-called trophic disturbances must be considered chiefly as vasomotor and angiosclerotic in nature. If this idea be accepted, we have perhaps an explanation of the heaping up of bony tissue in the joint in some cases of arthropathy as due to excess of nutrition from dilatation, and of wasting in other cases as due to contraction and angiosclerosis of the vessels.

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[1] Wollenberg: Archiv f. Psychiatrie, 1892, xxiv.

[2] Stroebe: Centralblatt für allgemein. Path. u. path. Anat., 1894, p. 853.

[3] Darkschewitsch: Archiv für Psych., 1892.

[4] Fraenkel: Wiener klin. Wochenschrift, 1896, Nos. 9 and 10.

While we would be loath in the light of our present knowledge to attribute all trophic changes to a vascular origin alone, it seems that a large part of them are due to alterations in the circulation as well as to loss of sensation in the part, the exposure of a paralyzed limb to injury, uncleanliness, and other similar causes. The few cases of chronic anterior poliomyelitis followed by autopsy which are recorded—not more than seven or eight as collected by Nonne[1]—and the recent case of pure motor neuritis reported by Dejerine and Sottas,[2] in which trophic changes other than atrophy were absent, indicate that trophic fibres, if they exist, are not to be found in the peripheral motor neurons. Cases of tabes and syringomyelia point to the sensory neurons as the location of these disturbances.

Such conditions as loss of teeth, perforating ulcers, arthropathies, fragility of the bones, glossy skin, vitiligo, herpes, pemphigus, brittleness of the nails, discoloration and loss or excessive growth of hair, etc., do not seem to be dependent on the motor neurons.

Schaffer[3] has recently found the motor cells of the lumbar region diseased in a case of tabes with arthropathy of the knee. He employed the method of Nissl. We cannot decide from his paper whether the cells on the side corresponding to the arthropathy were more involved in the degenerative processes or not; nor does it seem to us improbable that these slight changes in the motor cells were due to alteration in the reflex collaterals. It is not known how frequently they occur in cases of tabes without arthropathies. Possibly the axis-cylinders of some of these diseased cells were to be found in the posterior roots.

Schlesinger[4] has collected fifty-three cases of syringomyelia with arthropathy, of which eleven only were confirmed by autopsy. To this number he adds four cases of his own. This case of ours, according to his statistics, makes the sixteenth.

POSTERIOR ROOTS. Considerable confusion exists in regard to the condition of the posterior roots in syringomyelia, and the questions arise whether disease of the posterior roots can lead to syringomyelia, as combination of tabes with this affection is known, and whether destruction of a posterior horn can cause degeneration in the peripheral sensory fibres, even when the spinal ganglia are intact. According to Joseph,[5] fibres are found in the posterior roots which do not enter into relation with the cells of the spinal ganglia, whose trophic centres are in the cord, and v. Lenhossék,[1] Cajal,[2] and Van Gehuchten[3] have been able to demonstrate fibres which leave the cord through the posterior roots, at least in the chick. Retrograde degeneration must also be considered in this connection. Our case is in accord with those of Hoffmann, for even where the pachymeningitis is intense the posterior roots appear normal, as was observed also in a similar case of Gombault and Philippe,[4] and, although few or no fibres can be seen entering the right posterior horn in the cervical cord, the extramedullary portion of the roots is normal. There is no degeneration in the right column of Burdach in the cervical cord, as the fibres which enter into the formation of this column do not pass through the degenerated posterior horn.

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[1] Berliner klin. Wochenschrift, 1896, No. 10.

[2] Comptes Rendus des séances de la Soc. de Biologie, 1896.

[3] Revue Neurologique, Feb. 29, 1896.

[4] Die Syringomyelie.

[5] Joseph: Archiv für Anatomie und Physiologie, 1887.

LIMITATION TO ONE POSTERIOR HORN. Oppenheim[5] is authority for the statement (1893) that there are only two cases recorded of unilateral syringomyelia with corresponding symptoms and confirmation of the diagnosis by autopsy. These are the case of Rossolimo,[6] and the one of Dejerine and Sottas.[7]

Hatschek,[8] in June, 1895, could find no other recorded cases. One of his own presented unilateral symptoms with predominance of the lesion on one side. Oppenheim (1893) can report only one case, and that without autopsy, in which the syringomyelia was extensive in the lower dorsal cord and confined to one posterior horn throughout the cord above, as appeared to be the condition clinically in the case we report. In our case the gliosis extends through the lumbar and sacral regions, although the increase of the patellar reflexes would have indicated that these parts were not involved. It becomes limited to one posterior horn at the first dorsal segment, which explains the absence of all paralysis in the arm, although disturbance of thermal sense and some analgesia were present. We have been able to find some of the spinal lesions described in the case of Rossolimo. The fibres of the right posterior roots entering the right posterior horn are destroyed, the right anterior horn contains fewer fibres in the lower cervical region, and here also the right side of the cord is smaller than the left, noticed especially in the lateral column; but the number of ganglion-cells of the anterior horn is not lessened and the lemniscus is normal on the opposite side. In Rossolimo's case the posterior horn was affected from the tenth or eleventh thoracic roots through the cervical region; this greater extent of destruction may possibly explain the condition of the lemniscus

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[1] v. Lenhossék: Anat. Anz. 1890 Jahrg., v. pp. 360–362.

[2] Ramón y Cajal: Anat. Anz. 1890 Jahrg., v. p. 613.

[3] Van Gehuchten: Anat. Anz. 1893 Jahrg., viii. p. 215.

[4] Gombault and Philippe: loc. cit., p. 553.

[5] Oppenheim: Archiv f. Psych., vol. xxv. p. 317.

[6] Rossolimo: Ibid., vol. xxi.

[7] Dejerine and Sottas: Comptes Rendus des séances de la Société de Biologie, 1892.

[8] Loc. cit., pp. 1155 and 870.

of the opposite side in his case; and yet one would imagine that in a lesion from the first dorsal to the second cervical segment the extent would be sufficient to produce a similar alteration in the lemniscus. In his case he was unable to report the condition of the nuclei of the posterior columns, and these columns in the upper portion of the cord were not entirely free from sclerosis. The lateral column on the same side as the lesion was smaller than that of the other side, as is the condition also in the lower cervical portion in our case; but we must explain this in greater part as due to the ascending sclerosis. Fibres probably ascend in the antero-lateral column to join the lemniscus of the same side at the sensory decussation, but we are not aware of any connection between these and the lemniscus of the opposite side. The theory of Rossolimo, that the degeneration of the lemniscus in his case was due to a presumable alteration of the posterior nuclei, seems most probable.

We have in the microscopic examination a satisfactory explanation of the condition observed in life. The paralysis and contracture of the lower limbs, incontinence of the bladder and rectum, decubitus over the buttocks, absolute loss of cutaneous sensibility in both legs, constricting pains over the chest, are explained by the pachymeningitis and total destruction of the middle and lower portions of the thoracic cord. We find that tactile sensibility began to be normal at the level of the nipple, on the right side, and of the umbilicus on the left, and we have reported that at the second dorsal segment the cavity was almost limited to the right posterior horn; furthermore, it is stated that over the right arm, right shoulder, right side of the neck, the patient failed to recognize the difference between heat and cold, and that some analgesia was present over the right arm. Does not the limitation of the cavity to the right posterior horn prove beyond question the location of fibres for the sense of temperature for a portion of their course within the posterior horns?

A zone of tissue between the extreme median end of the cavity and the central canal was not destroyed; it is possible that fibres for the sensation of pain may be located here, and that in this way the partial analgesia may be explained. Our case shows further that the thermal fibres enter the posterior horn to the outer side of that portion which in the thoracic cord is occupied by the column of Clarke.

In Rossolimo's case we find mention of complete loss of temperature- and pain-sense in the upper extremity corresponding to the side of the cavity. The preservation of tactile sense in the right upper limb, and the presence of a cavity which destroyed the right posterior horn throughout the cervical region, indicate that tactile fibres from the upper extremity probably ascend in the column of Burdach.